

Background

Because CWD is relatively new, much remains unknown about the disease, how it functions, its epidemiology, and transmission dynamics. Intensive research on CWD and how it affects cervid populations has only begun in the last decade. This chapter describes: 1) what is known about CWD at this time; 2) what is currently being done at the national and state levels to combat CWD; 3) the history of CWD in Wisconsin; and 4) what actions Wisconsin state agencies have taken up to this point in time to control the disease. This information was used to develop the CWD Management Plan and proposed rule assessed in this document.

Description of Disease.

CWD belongs to the family of diseases known as transmissible spongiform encephalopathies (TSEs), so called because these diseases cause microscopic holes in brain tissues giving it a sponge-like appearance. TSEs include diseases such as scrapie in sheep, bovine spongiform encephalopathy (BSE) in cattle, and Creutzfeldt-Jakob disease of humans. The causative agent is believed to be a protease-resistant protein (or prion). These modified proteins are typically found in nervous and lymphatic tissues, but recently prions have been detected in muscle tissue of mice experimentally inoculated with BSE prion. Prions have not been detected in muscle tissue of naturally infected species, although research on prion diseases is unusually difficult because prions are difficult to detect.

No treatment is known, and the disease is typically fatal in cervids. Infected deer and elk can appear robust and healthy in the early stages of CWD and may take two or more years before they show clinical signs of the disease. The clinical signs are not unique to the disease and can be due to other conditions such as malnutrition. Currently, the “gold standard” or nationally recognized test for diagnosing CWD requires the microscopic examination and/or immunohistochemistry (IHC) staining of a specific portion (obex) of the brain. In addition, IHC tests for retropharyngeal lymph nodes (found in the head) can indicate early (4-6 months from the time of first exposure) CWD infection in deer (Wolfe *et al.* 2002, Wild *et al.* 2002).

The spread of CWD in wild animals is of great concern. The disease was originally described in captive animals 35 years ago in Colorado. CWD was first detected in free-ranging mule deer in 1981; however, epidemiological modeling suggests that CWD may have been present in Colorado and Wyoming for 15-20 years before it was discovered in free-ranging deer (Miller *et al.* 2000). Over the last five years, however, CWD has been found in wild herds in several surrounding states and Canada (Figure 1). In 2002, CWD was detected in wild deer in South Dakota, New Mexico, Wisconsin, and most recently, Illinois. The recent detection of CWD in the wild white-tailed deer herd in Wisconsin is of particular concern due to the potential for rapid spread within white-tailed deer populations of substantially higher densities than in the previously identified endemic areas of Colorado, Wyoming, and Nebraska (2-5 deer per square mile). Fall deer densities in south-central Wisconsin can be as high as 75+ animals per square mile.

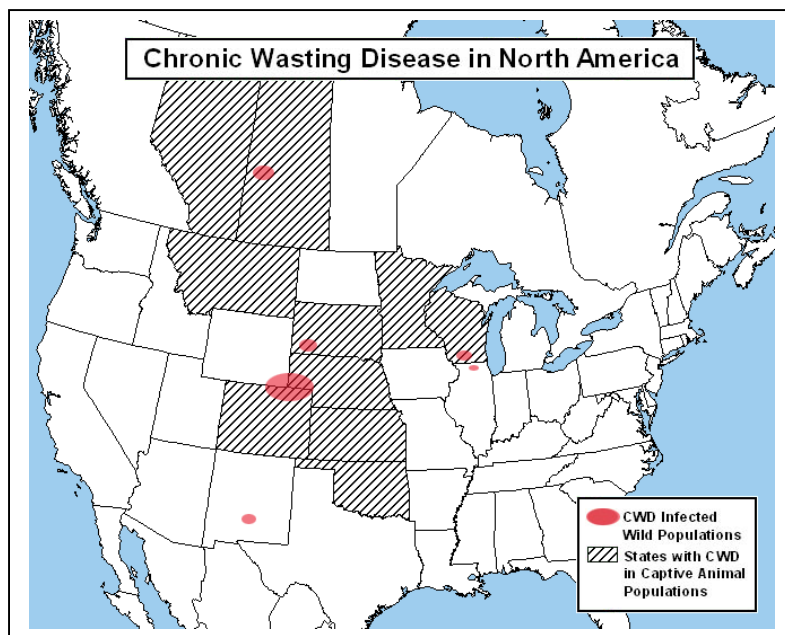


Figure 1. Current known distribution of CWD in free-ranging and captive deer and elk populations.

Agent.

The agent causing CWD and other TSEs is incompletely characterized, but it appears to be a prion. Prions are proteins that are naturally produced in nerve, lymphoid, and the cells of other tissues. Most data support the hypothesis that the causative agent of CWD is an abnormal, protease-resistant form of the normal prion.

In humans, abnormal prions seem to arise most commonly via sporadic mutations or spontaneous conversion of normal proteins to abnormal prions, although there seem to be genetic and age factors related to susceptibility in humans. In animals, TSEs can be transmitted as food-borne diseases associated with animal products and can function like infectious agents. Spontaneous forms have not been identified. It is possible that abnormal prions resulted from spontaneous alteration of normal prions to abnormal prions with subsequent transmission to susceptible deer and elk. Alternatively, CWD could be a strain of scrapie that has adapted to cervids (Race *et al.* in review). Additional, though relatively weak, evidence for a link between scrapie and CWD is the moderate ability of abnormal prions to convert bovine normal prions *in vitro* (Raymond *et al.* 2000) and the susceptibility of goats to intracerebral exposure to the CWD agent (Williams and Young 1992). CWD also could have originated by infection with an unidentified prion strain. The CWD agent differs from the BSE agent, many strains of scrapie, and transmissible mink encephalopathy agent based on mouse strain typing and molecular (glycoform) pattern comparisons (Bruce *et al.* 1997, 2000; Race *et al.*, in review). The marked similarity of central nervous system lesions, epidemiology, and glycoform patterns strongly suggests the CWD agent is the same in farmed, captive, and free-ranging deer and elk (Williams and Young 1993, Spraker *et al.* 2002). Whether multiple strains of these abnormal, protease-resistant prions occur in nature remains under study (Williams *et al.* 2002a).

Distinct prion strains (for TSEs) with specific host affinities, pathotypes, and molecular profiles have been recognized from studies of infected animals or tissues (Bruce *et al.* 1994, 1997, 2000; Raymond *et al.* 1997, 2000; Safar *et al.* 1998, 2000; Race *et al.*, in review). The nature of these strain differences remains controversial and is still under investigation (Williams *et al.* 2002a). Strain typing experiments determined that CWD is not like known scrapie strains (Bruce *et al.* 2000), though direct comparisons with North American strains have not been conducted.

Susceptibility.

Mule deer, white-tailed deer, and Rocky Mountain elk are known to be naturally susceptible to CWD. . . . Both sexes and all age classes show relatively uniform susceptibility (Miller *et al.* 2000, Williams *et al.* 2002a). In contrast, a variety of wild and domestic species appear to be resistant, or at least much less susceptible to CWD, although the numbers of animals tested remains small. So far, moose, pronghorn antelope, Rocky Mountain bighorn sheep, mouflon, mountain goats, and blackbuck held in contact with CWD-affected deer and elk or resident in premises where CWD occurred have not developed the disease. Domestic livestock are not known to be naturally susceptible to CWD. A few cattle, sheep, and goats have resided in research facilities with CWD for prolonged periods without developing the disease. Cattle intensively exposed to CWD-infected deer and elk via oral inoculation or confinement with infected captive mule deer and elk have remained healthy for over five years (Williams *et al.* 2002b). These observations of apparent species barriers to efficient transmission are supported by molecular and intracerebral challenge studies (Raymond *et al.* 2000; Hamir *et al.* 2001; Williams and Young, unpubl. data).

Many species can be experimentally infected with CWD (and other TSEs) when exposed via intracerebral inoculation, an unnatural route of exposure commonly used to study prion diseases (Williams *et al.* 2002b). Mink, domestic ferret, squirrel monkey, mule deer, domestic goat, domestic cattle, and lab mice have been infected with CWD by this route (Williams and Young 1992; Bartz *et al.* 1998; Bruce *et al.* 1997, 2000; Hamir *et al.* 2001; Marsh, Young, and Williams, unpubl. data).

There is no known antibody response to the CWD agent (Williams *et al.* 2002a), although antibodies have been reported to offer resistance to disease in laboratory studies. Specific amino acids (Codon 132 methionine) were over-represented among free-ranging and farmed CWD-affected elk compared with unaffected elk (O'Rourke *et al.* 1999), indicating the potential for differential susceptibility. The vast majority of captive deer residing in endemic research facilities eventually contract CWD, but some

individuals occasionally survive a lifetime without succumbing to CWD (Williams *et al.* 2002a). A specific genotype (PNRP), which plays a major role in the development of scrapie in sheep (Hunter *et al.* 1992, Bruce *et al.* 1994, O'Rourke *et al.* 1997), has not been demonstrated in deer, but remains under investigation (Williams *et al.* 2002a).

It has been suggested that some deer may possess a genetic resistance to CWD and that the fatal brain disease could be controlled by genetically improving the deer herd. Others have suggested that genetically engineering a CWD resistant deer may be possible. Current scientific evidence indicates that the vast majority of white-tailed deer are likely susceptible to CWD, and there is no evidence confirming genetic resistance in this species. While a genetic strategy may appear theoretically viable, there is no practical evidence to suggest that free-ranging deer could be manipulated by genetic engineering, artificial breeding, or propagation and release of animals in order to produce a genetically resistant population. Considering the long course of disease in deer, the high annual reproductive potential of females, and the limited ability to control breeding in free-ranging deer, strategies to manipulate genetic resistance of white-tailed deer to CWD is not currently practical.

Another theory suggests copper deficiency in deer and elk may be linked to CWD. Copper is a naturally occurring mineral vital to proper brain functions. Deer and elk may become copper deficient when they live on crowded range where copper sources found mostly in natural browse are depleted. The basic argument is that copper deficiency may increase the susceptibility of deer and elk to CWD. It is important to note that although copper deficiencies have been linked to other diseases, there is no proven link to CWD susceptibility in wild deer and elk. Additional research is needed to determine if copper plays a role in the susceptibility of deer and elk to CWD.

Transmission and Routes of Infection.

CWD is both transmissible and infectious, but specific details regarding transmission remain unknown (Williams *et al.* 2002a). In contrast to BSE, CWD does not appear to be exclusively a food-borne disease associated with rendered ruminant meat, bone meal, or animal protein products. Contact between infected and non-infected animals via saliva, urine, and feces is the most likely route of transmission. Data from CWD epidemics in captive cervids (Williams *et al.* 2002a) and field data from wild cervids provide strong evidence that lateral transmission (animal-to-animal) is the primary form of infection in susceptible animals. Computer simulations indicate that vertical transmission (in utero mother-to-offspring), if it occurs, is unlikely to maintain CWD in a deer population (Miller *et al.* 1998, 2000). Transmission via contact between susceptible and infectious individuals probably requires more than just transient exposure. The concentration of deer and elk in captivity or by artificial feeding probably increases the likelihood of direct transmission among individuals (Williams *et al.* 2002a). Interspecific transmission probably occurs among the three susceptible native cervid species (mule deer, white-tailed deer, and elk) when their ranges overlap.

Because prions are resistant to degradation in the environment (Brown and Gajdusek 1991), indirect transmission via contamination of the environment by excreta (urine and feces) from infected animals is possible. Contaminated pens and pastures may have served as sources of infection in captive cervids, (Miller *et al.* 1998, Williams *et al.* 2002a). However, CWD apparently did not persist in several facilities that experienced a few cases of CWD and presumably were not heavily contaminated. The apparent persistence of abnormal prion in contaminated environments represents a significant potential obstacle to eradication of CWD from farmed cervid populations. The importance of environmental contamination in free-ranging animals is not clearly understood.

Based on available information, the brain, spinal cord, and the lymphoid tissues of the gut and head are likely to be the greatest source of infectious material (Spraker *et al.* 2002) (Figure 2). The brain and spinal cord, by virtue of its mass and high titer, and the lymphoid tissues of the gut and head, because of its access to the external environment, are of concern. Based on histology and immunohistochemistry (IHC), the abnormal prion deposition has also been detected in the pituitary and adrenal glands as well as the pancreas (Sigurdson *et al.* 2001).

Because of the possibility of indirect transmission of CWD, the best methods for disposal of unwanted carcasses, parts of carcasses, or disinfecting tools used on CWD infected animals remain unclear. An abnormal prion is a highly resistant material that can retain infectivity under a wide range of conditions including treatment by heat (boiling and dry), irradiation, and most conventional disinfectants. Therefore, the choice of disposal method for CWD-infected and potentially infected carcasses and tissues and disinfecting tools used to butcher deer becomes an important concern or perceived concern for contaminating the environment. Environmental contamination with prions could possibly affect the health of free-living deer populations. Four methods of disposal have been suggested: landfills, burning at temperatures over 1200° F, rendering, and chemical digestion. Which method is best to contain and destroy the prions is unclear. A detailed discussion of these methods can be found in Appendix C.

Pathobiology.

Experimental CWD challenge studies based on single-dose, oral infections have provided some insight into timing of the course of the disease. However, these conclusions are limited, because the course of infection may be inversely related to the exposure dose (Williams *et al.* 2002a). Experimentally, the minimum latency period (time from exposure to onset of clinical signs) was about 15 months and the average time to death was about 23 months (range 20 to 25 months) in mule deer (Williams *et al.* 2002a). The range of latency period in orally infected elk was approximately 12 to 34 months (Williams *et al.* 2002a). Duration is uncertain, but likely to be shorter in wild cervids. Among deer and elk residing in facilities with a long history of CWD, most natural cases occur in 2-7 year old animals, but some deer have survived longer than seven years in heavily infected facilities.

Although prion biochemistry is not well understood, the following is a likely route of prion amplification and transmission through animal systems (Figure 2). In natural cases, the route of infection is believed to be oral. In the gut, prions may be taken up by the lymphoid tissues (Peyer's patches), then transported around the body in lymphocytes. Nerves that supply the lymphoid tissues then allow prions to travel back up these tissues to the brain. Since lymphocytes carry prions, it is reasonable to assume that transport to other lymphoid tissues might occur in parallel through the blood. Prion replication is believed to occur in the lymphoid tissues, especially the spleen. When normal prions encounter CWD abnormal prions, the abnormal prions are thought to force the normal prions to change shape (both prions are the same amino acid sequences, but have different shapes). This shape-flipping is equivalent to the transmission of infection. The abnormal prions are resistant to degradation by the natural cellular protease enzymes, therefore, abnormal prions accumulate in the cell.

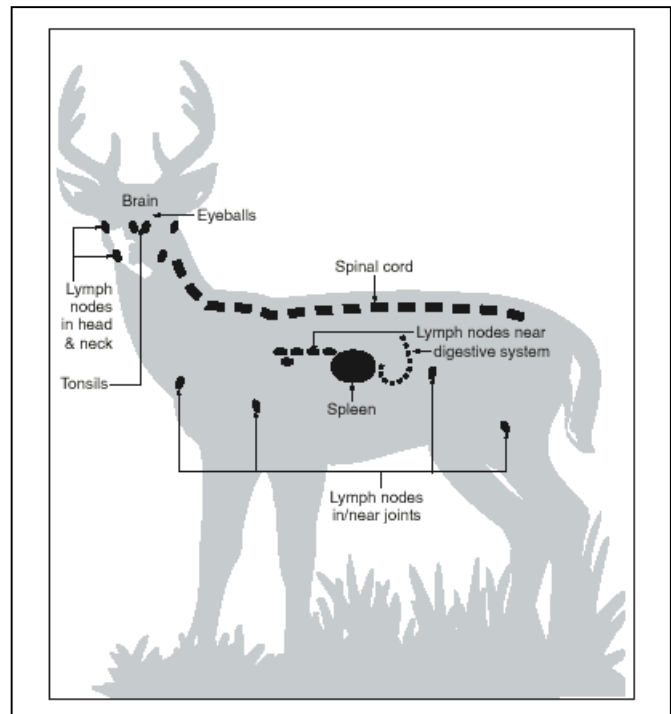


Figure 2. Location of lymph nodes and organs where concentration of CWD prions are located.

It is not known when during the course of infection an animal becomes infectious, but abnormal prion shedding may be progressive through the course of disease in deer (Williams *et al.* 2002a). Accumulation of abnormal prions in gut-associated lymphoid tissues (*e.g.*, tonsils, Peyer's patches, lymph nodes) early in the latency period suggests that shedding through the alimentary tract via feces and saliva may occur (Sigurdson *et al.* 1999, Miller and Williams 2002, Spraker *et al.* 2002). Epidemiological models for elk and mule deer suggest shedding probably precedes onset of clinical disease. Demonstration of abnormal prions in lymph nodes and tonsils of deer early in incubation (Sigurdson *et al.* 1999, Wild *et al.* 2002) provides a reliable means of

antemortem and preclinical diagnosis of CWD (Williams *et al.* 2002b). However, due to differences in pathogenesis of CWD in elk, sampling lymphoid tissue, such as tonsils in elk, does not appear to be sensitive enough to use as a reliable live diagnostic test (Williams *et al.* 2002b).

Animals that have clinical disease exhibit behavioral changes and progressive loss of body condition that invariably lead to death. Affected animals may walk repetitive courses, and may show subtle ataxia and wide-based stance. Subtle head tremors may occur. Affected animals may be found near water sources or in riparian areas, have periods of drowsiness, and carry their head and ears lowered. Affected animals continue to feed, but amounts of food consumed and/or digested are reduced, leading to gradual loss of body condition. Excessive drinking and urination are common in the terminal stages and many animals have excessive salivation and drooling, causing wetting of the hairs on the chin and neck (Williams *et al.* 2002b). Once the signs of CWD appear, the clinical course can vary from a few days to about a year, with most animals surviving from a few weeks to 3-4 months (Williams *et al.* 2002a). Clinical courses in wild cervids probably are shorter than in captive animals, because wild cervids must forage, find water, and are susceptible to predation. Aspiration pneumonia is a common finding at postmortem examination of terminal CWD cases. This condition presumably is due to difficulty swallowing, hypersalivation, and inhalation of foreign material into the lungs (Williams *et al.* 2002b). Clinical signs of CWD alone are not diagnostic, and definitive diagnosis is based on examination of the brain for spongiform lesions and/or accumulation of abnormal prion protein in the brain and lymphoid tissues by immunohistochemistry.

Prevalence.

CWD can reach high prevalence in captive cervids (Williams *et al.* 2002a). In one facility, greater than 90% of the mule deer exposed for greater than two years either died or were euthanized due to clinical CWD (Williams and Young 1980). Recently, CWD prevalence greater than 50% has been found in white-tailed deer confined in association with an infected elk farm (Nebraska Game and Parks Commission 2002). Among captive elk, CWD was the primary cause of adult mortality in two research herds (Miller *et al.* 1998), and high prevalence has been reported from other farmed herds (Williams *et al.* 2002a).

Based on tissues from animals collected by hunters in the CWD-endemic area in Colorado, prevalence of pre-clinical CWD was estimated from approximately 1-15% in mule deer, 1-22 % in white-tailed deer, and less than 1 % in elk (Miller *et al.* 2000). In Wisconsin, prevalence of CWD in white-tailed deer was estimated at approximately 3 % in a newly discovered affected area, but local prevalence at the center of the area was approximately 13% from data collected in April 2002.

Prevalence was similar between male and female mule deer (5.5% vs. 3.6%), white-tailed deer (2.3% vs. 1.4%), and elk (0.7% vs. 0.3%) in the endemic area of Colorado (Miller *et al.* 2000). For mule deer, prevalence differed between age classes within each sex. Prevalence was consistent across age class for females, but increased through the 4-6 year class then declined in males (Miller *et al.* 2000).). In Wisconsin, prevalence of CWD in white-tailed deer sampled in the spring and summer of 2002 did not vary among males and females (males: 2.8%, 95% confidence interval 1.1-5.7%, n = 250; females: 2.6%, 95% confidence interval 1.6-4.0%, n = 795), nor did prevalence change with age in the spring 2002 sample (odds ratio 1.13, 95% CI 0.93-1.39, n = 476).

Factors Affecting Changes in Prevalence and Geographic Spread of CWD.

Little is known about the rate of increase in prevalence and geographic spread of CWD or the factors that affect these rates. CWD was first detected in free-ranging mule deer in 1981, however, epidemiological modeling suggests that CWD may have been present in Colorado and Wyoming for 15-20 years before it was discovered in free-ranging deer (Miller *et al.* 2000). Surveillance in free-ranging mule deer populations did not begin until the mid-1990s. By 1999, the known endemic area of Colorado and Wyoming covered 15,000 square miles (Miller *et al.* 2000). Prevalence in mule deer within the endemic area varied among geographically distinct subpopulations from less than one percent to nearly 15%.

Increases in CWD prevalence in Colorado and Wyoming have been relatively slow. Epidemiological modeling suggests that prevalence in Colorado and Wyoming may have increased 0.5% to 0.7% annually during the 1980s and 1990s. Miller *et al.* (2000) characterized CWD in Colorado and Wyoming as an "epidemic with a protracted time scale". Simulation modeling, however, suggests that small changes in

transmission rates can greatly affect the rate of change in CWD prevalence. Gross and Miller (2001) proposed that the rate of change in CWD prevalence is affected by 1) the rate of disease progression (incubation period and clinical course) within infected individuals, 2) the rate of CWD transmission between infected and susceptible individuals, and 3) the rate of population turnover (a function of reproduction and mortality). Miller *et al.* (2000) estimated 1.2-1.3 new infections per infectious animal per year for mule deer populations. These estimates were from relative low-density deer populations in Colorado and assume random, density-independent transmission among individuals. However, Miller *et al.* (2000) indicated that transmission rates would need to be much higher (about 3.5 new infections per infectious animal per year) to simulate epidemics in captive deer populations where extremely high prevalence (50-90%) has been observed. These results suggest more intensive transmission under confinement or in high-density populations. It is not known whether transmission rates are dependent on wild deer density and no studies have been conducted to directly measure CWD transmission among wild cervids. Mule deer densities in northeast Colorado and southeast Wyoming are much lower than white-tailed deer densities in southern Wisconsin. In addition, the deer populations in southern Wisconsin are more contiguous than the geographically distinct populations of mule deer in the Colorado and Wyoming endemic areas. The substantial difference in deer density and distribution of populations between these regions raises concerns that transmission rates may be higher in Wisconsin than in western mule deer. In addition, factors that influence CWD transmission rates are poorly understood, but likely there is a complex relationship between CWD transmission, the social organization and density of the cervid species, and habitat-related differences in dispersal and movement patterns.

Currently there are no data available to link animal densities to prevalence rates in wild cervids. Colorado is currently testing whether population density reduction results in reduced prevalence (Ver Steeg 2002). The density of deer in Wisconsin's CWD management areas may be as much as 10 times that of the density found in western free-ranging populations where CWD is endemic prompting concern about rapid spread of CWD. Because CWD is readily transmitted among captive cervids, it is believed that transmission may also be facilitated by the concentration of animals due to artificial baiting and feeding.

Deer Ecology as it Relates to CWD.

The discovery of CWD in southwestern Wisconsin represents the first instance of the disease in a high-density white-tailed deer population. Simulation modeling suggests that the rate of change in CWD prevalence can be greatly affected by small changes in transmission rates (Gross and Miller 2001). Factors that influence transmission rates are poorly understood, but transmission rates are likely affected by deer density and by species and habitat-specific differences in movement patterns and social structure of deer populations. In addition, deer ecology also affects the population consequences of CWD and the ability to manage the disease. Rates of reproduction, natural and human-caused mortality, and dispersal for white-tailed deer in southwest Wisconsin are likely very different from the published results of Rocky Mountain mule deer.

Movements.

Although uncertainty remains about the mechanism of CWD spread across landscapes, it is generally believed that dispersing deer are a likely avenue of disease spread. Male and female fawns generally remain with their mother through their first year of life. Female fawns often remain in the same social group as their mother. These matrilineal family groups consist of a dominant female, her female offspring, and their fawns (Mathews 1993, Aycrigg and Porter 1997). Matrilines tend to be philopatric, traveling together throughout most of the year (Hirth 1977). Prior to fawning, matrilines tend to separate such that each female occupies largely exclusive fawning ranges configured in a "rose-petal" arrangement with the dominant females at the center and subordinate females surrounding (Ozoga and Verme 1982). However, lack of available fawning habitat or disruption of the matrilines may result in female dispersal, especially among one and two-year olds (Nixon *et al.* 1991).

In contrast, male fawns usually disperse from their natal ranges as they approach puberty (12-18 months of age; Nelson 1993). Dispersing males often form social groups of unrelated individuals. These male groups separate during the breeding season, and breeding territories are established and defended by

males that are mature enough or aggressive enough to assert local dominance. Sub-dominant males may show a range of breeding season behaviors from essentially non-participation to “floater” and interloper behaviors in and around established territories (Ozoga and Verme 1982).

Dispersal distances in the Midwest can be as long as 130 miles (Kernohan *et al.* 1994) although analysis of dispersal distance distributions (Nelson 1993) suggests that long-distance (greater than 30 miles) are quite rare and that median dispersal distances are more likely in the 5-10 mile range (Nelson 1993). If specific habitats are limited (*e.g.*, forest in the intensively-row-cropped corn belt), median dispersal distances can be much larger (about 25 miles; Nixon *et al.* 1991). In southern Wisconsin, a total of 197 does and 87 bucks were marked during four studies conducted in the 1970s and early 1980s (Ishmael 1984, Wozencraft 1978, O'Brien 1976, Larson 1974). Movements were noted for six does and 12 bucks. The maximum distances recorded in these four studies ranged from 6-12 miles and mean distances were 3-4 miles. Etter *et al.* (2002) reported mean dispersal distances of 3-5 miles for doe and buck fawns in a study in the Chicago, Illinois suburban forest preserves.

Reproduction and Mortality.

White-tailed deer in southern Wisconsin have relatively high rates of reproduction and mortality, resulting in fairly rapid population turnover. Midwestern habitats generally have lush temperate vegetation that provides ample forage and secure hiding places. As a result, female deer typically maintain a high nutritional plane, have high rates of reproduction, and survival of fawns is relatively high. Pregnancy rates in southern Wisconsin during the mid-1980s were 51% for fawns, 88% for yearlings, and 95% for adult does. Mean in utero litter sizes were 1.15 for fawns, 1.66 for yearlings and 1.90 for adult does (McCaffery *et al.* 1998). During the 1990s observed fawn:doe ratios during July-September averaged 1.06 in southwestern Wisconsin (Wisconsin DNR, unpubl. data). These reproductive parameters result in an average annual rate of increase of 1.4 between midwinter and the subsequent fall, meaning the population, on average, will increase by at least 40% per year from the addition of fawns.

Deer populations in southwestern Wisconsin are heavily hunted with average densities of more than 20 hunters per square mile of deer habitat on opening day of the November gun season. Estimates of adult survival rates from demographic models for southern Wisconsin average about 30% for males and 60% for females during the late 1990s (Wisconsin DNR, unpubl. data). The difference in adult survival rates between sexes has an overwhelming influence on the age and sex structure of the population.

Estimates of non-hunting mortality are not available for southwestern Wisconsin, but this type of mortality is believed to be minor. Coyotes can be an important source of neonatal mortality in areas associated with high deer density and limited fawning habitat (Nixon *et al.* 1991, Piccolo 2001), however, high late summer fawn:doe ratios in southwestern Wisconsin suggest that fawn survival is good. Deer-vehicle accidents are numerous in Wisconsin, but consequent population effects in rural areas are usually minor (Nixon *et al.* 1991, Van Deelen *et al.* 1997). Weather-related starvation occurs infrequently in southern Wisconsin (Wisconsin DNR 2001). Previously, disease has not been a significant source of deer mortality in Wisconsin. CWD and epizootic hemorrhagic disease (EHD), a viral disease transmitted by biting midges, have only recently been found in Wisconsin's wild deer herd. However EHD is seasonal, usually occurring in late summer and early fall, and ending when frost and cold weather kills the midges.

Wildlife Disease Control.

Goals for managing wildlife diseases include: 1) preventing the introduction of disease; 2) controlling the spread of the existing disease; or 3) eradication of the disease (Wobeser 2002). Strategies for disease management include directly attacking the disease agent, blocking the transmission among individuals, managing environmental conditions to reduce transmission, or reducing the population of infected and susceptible individuals below the threshold required for the disease to persist.

Wobeser (1994) reviewed the use of population reduction as a technique for controlling disease in wild animals. This method is based on the premise that infectious disease is a density-dependent process (*i.e.*, host population density is an important factor affecting the rate of transmission of contagious diseases). For many diseases there is a minimum threshold level of the host population below which transmission does not occur. However, because the transmission process differs for each disease the threshold level is

different for every disease situation. The rate of disease transmission to susceptible hosts may depend on the contact rate among hosts, number of susceptible hosts, and the number of infected hosts. In addition, the social behavior of a wildlife species can influence the transmission of disease. Options for host population reduction include selective removal of diseased animals, general reduction of population density, and elimination of the total population that may have been exposed to the disease.

Depopulation has been used to control a variety of diseases including rabies, plague, avian cholera, tuberculosis, histoplasmosis, rinderpest, brucellosis, and foot-and-mouth disease. Depopulation has been used most frequently for diseases of carnivores, bats, rodents, and occasionally for game species. This likely reflects public perceptions and concern about the desirability of killing certain species. Population reduction, however, is being used as one of several strategies to control tuberculosis in white-tailed deer in Michigan (Thorne *et al.* 2000).

Population reduction is a race between the removal of animals from the population by all causes, including the reduction effort, and recruitment through reproduction and immigration from the adjacent area. The probability that a population reduction program will be successful is dependent upon the intensity, consistency, and duration of the control effort and the specifics of the disease-host relationship including the transmission process, duration of infectivity, and the host species reproductive rate and dispersal tendencies. Population reduction requires continued effort over a lengthy period of time to be effective due to the potential for population growth via reproduction and ingress.

Host population reductions can be classified as: 1) focal depopulation about a specific site; 2) depopulation of an area surrounding the disease to create a barrier; and 3) general depopulation over a large area. Depopulation has been more effective in preventing entry of a disease into an area than in trying to control an established disease. It has also been more effective in dealing with isolated areas of disease than with a widely distributed disease. Consequently, control efforts have tended to de-emphasize extensive population reduction over large areas in favor of intensive control on smaller areas. In addition, there has been a greater use of specific information about the ecology of both the host and the disease agent in planning the program. The success of local or barrier depopulation is dependent on effective disease surveillance in order for control measures to be applied promptly in the correct location. Delay in recognition or reporting may allow the disease to spread widely making it more difficult to control.

Although population reduction has been used in an attempt to manage many diseases, the degree of population reduction achieved and the success of such programs has been highly variable. It is difficult to assess the effectiveness of many population reduction efforts because few such programs have objectively evaluated the effect of the depopulation on disease control. Only subjective assessments of effectiveness are available for most depopulation programs.

The culling of over 22,000 deer in California in 1924 was successful in eradicating foot-and-mouth disease (Fletcher 1970). Depopulation of wildlife species was an important component in efforts to control rinderpest epidemics in southern Africa during the 1910s and 1930s (Scott 1981). In contrast, host population reductions have been less successful in efforts to control myxomatosis in rabbits (Yuill 1981) and sylvatic plague in rodents (Olson 1981).

Early efforts to control rabies in vampire bats by destruction of bats at roosts were largely unsuccessful but development of more sophisticated methods of killing bats with poisons greatly increased the effectiveness of control efforts. Control efforts for vampire bat rabies have also benefited from increased knowledge of disease epizootiology and bat behavior that facilitated more precise geographic targeting of control activities. A 95% reduction of the vampire bat population in advance of a rabies epizootic in Argentina was successful in blocking the spread of the disease (Wobeser 1994). Vampire bat control efforts in Brazil and Venezuela were also successful in stopping outbreaks.

A massive depopulation of foxes, coyotes, wolves, and other carnivores was conducted in Alberta during 1952-54 in an effort to stop an epizootic of rabies. The program was viewed by some as successful due to the apparent eradication of the disease. However, the same epizootic diminished in other Canadian provinces at about the same time without similar depopulation programs (MacInnis 1987).

The effectiveness of depopulation of foxes for controlling a rabies epizootic in Europe is questionable. Gassing of dens and shooting for bounty during the 1960s was estimated to reduce the fox population by 80% in a control zone, limiting the spread of rabies into Denmark and eventually eliminating it. Similar efforts were successful in preventing a rabies epizootic from entering a disease-free area of Switzerland. Both of these efforts benefited from geographic barriers that helped limit movement of the disease. In contrast, fox rabies control efforts in other parts of Europe apparently were ineffective (MacDonald and Voigt 1985), likely due to the high reproductive rate in good quality habitat and long dispersal distances. These host biological characteristics necessitate a high level of effort to maintain areas free of foxes (Anderson *et al.* 1981).

During the early 1970s, Alberta used depopulation in an effort to prevent skunk rabies from entering the province. Poisoning, shooting, and trapping were used in a 30 km x 635 km zone along the border of Saskatchewan. In addition, depopulation activities were conducted within five km of all reported rabid skunks outside of the border zone. The program was judged to be successful in reducing the spread of rabies but it did not totally prevent occurrences of rabies in Alberta. Subsequent rabies outbreaks in Alberta were effectively contained by intensive skunk control (Rosatte *et al.* 1986). In contrast, limited control efforts in Saskatchewan and inconsistent efforts in Montana appeared to allow rabies to increase in prevalence and spread (Pybus 1988).

Total extirpation of host species from an area can be extremely difficult because, as populations are reduced, the effort required for further reduction increases substantially. Therefore, eliminating the last few individuals becomes very difficult. The Brucellosis and Tuberculosis Eradication Campaign in Australia significantly reduced populations of feral water buffalo (Freeland and Boulton 1990). On a 389 square km area, the buffalo population was reduced to less than 1 % of its initial size by a combination of helicopter roundup and ground and aerial shooting (Ridpath and Waithman 1988). However, as buffalo density decreased, the time required to remove an animal by helicopter shooting increased substantially, in part due to behavioral changes (hiding and reduced diurnal activity) of buffalo subjected to aerial gunning over extended periods. Ridpath and Waithman (1988) concluded that eradication of buffalo from their entire range was an unrealistic objective. The time required to kill feral pigs in control programs in Australia increased exponentially as aerial shooting reduced population density (Choquenot *et al.* 1999). Saunders and Bryant (1988) felt that complete eradication of feral pigs during a disease outbreak might be unachievable. However, complete eradication of the host species may not be necessary to eliminate the disease. Disease elimination can be achieved if the host population is reduced below the transmission threshold.

Current CWD Management Plans.

Options for management of CWD are limited because no vaccine is available to prevent infection in susceptible animals and there are no known treatments for infected individuals. The long incubation period, possible environmental contamination with a persistent pathogen, and an incomplete understanding of the routes of transmission limits options for control of CWD (Williams *et al.* 2002a).

Management plans for CWD should be viewed as experimental and adaptive. Management plans should integrate research, management, and surveillance programs. They should be flexible and be altered using new knowledge gained from surveillance testing, research, and monitoring the effectiveness of the management program.

Management options for captive cervids are currently limited to quarantine or depopulation. Attempts to eradicate CWD from captive cervid farms by depopulation are currently underway in Colorado, South Dakota, and Wisconsin, however, it is too early to know if these efforts will be successful. Efforts to eradicate CWD from two captive research facilities in Colorado and Wyoming during the 1980s were unsuccessful. The reasons for these failures are not known, but a possible explanation may be contamination of the facilities with the pathogen and subsequent infection of reintroduced animals (Miller *et al.* 1998, Williams and Young 1992).

Management of CWD in free-ranging deer and elk populations will be considerably more difficult than in captive herds. Active surveillance programs to detect CWD in new areas, determine the spatial extent of CWD, to monitor changes in the prevalence of CWD over time, and to assess the effects of management actions are essential for successful management (Williams *et al.* 2002a).

The following section includes examples of proposed actions to control CWD that were developed by a national CWD management team for the country and by states and provinces where the disease has been found.

National CWD Management Plan.

During summer 2002, a National CWD Task Force developed a "Plan for Assisting States, Federal Agencies, and Tribes in Managing CWD in Wild and Captive Cervids". The task force was formed to ensure that federal and state agencies cooperate in the development and implementation of an effective national CWD program. It was composed of representatives from the U. S. Department of Agriculture, U. S. Department of Interior, numerous state wildlife and agricultural agencies, and universities. The task force concluded that there are many aspects of the ecology of this disease about which little is known and as a consequence management of CWD must be experimental and adaptive. Management must integrate research, management, and surveillance programs to enhance the capability to control this disease. The best available scientific information should be used to plan management actions, and these programs must include methods for assessing the effectiveness of each aspect of the management plan. As new information becomes available, and as results of intervention activities are assessed, management techniques should be adjusted or replaced accordingly. The effectiveness of management can only be assessed based on changes in distribution and prevalence of the disease. For this reason, rigorous ongoing surveillance must be an integral part of any CWD management plan.

The task force developed action plans in six areas: public communications, dissemination of scientific and technical information, diagnostics, disease management, research, and surveillance. The goal of the task force that developed the disease management action plan was to identify alternative practices for herd management that can help prevent the introduction of CWD into new areas, to eliminate CWD where it presently occurs, and to reduce the impact of CWD on wild cervids by reducing disease prevalence.

The Disease Management Working Group concluded that elimination of CWD is most feasible in captive populations, and the USDA and states have proposed a program to accomplish that goal. The Working Group felt that CWD elimination in the wild is most feasible with early detection of new disease areas. With new areas, there may be time to prevent significant disease transmission, reduce movement of infected animals, and minimize environmental contamination. The Working Group recommended that if CWD elimination is not possible, the management goal should be to control and limit spread of the disease. Control and limitation of the disease's spread is most appropriate in endemic areas where the disease has maintained itself for many years over a large area.

The Working Group believed the key to the management of this disease is the development of coordinated, science-based CWD plans tailored individually to the specific situation. The Working Group recognized that CWD management plans will vary depending upon such factors as length of time the disease has been present, affected species, population density, location, resources, and human dynamics.

The Disease Management Working Group identified a number of potential actions that could be components of a comprehensive management plan. These actions included disease prevention, disease and population management, captive cervid management, carcass disposal, monitoring and adaptive management, environmental decontamination, and population restoration.

In areas where CWD has been identified, the Disease Management Working Group recognized that there are a variety of management techniques to eliminate, contain, or control CWD. Outbreak surveillance establishes the prevalence, incidence, and distribution of the disease, and allows the evaluation of management actions. Population reduction can be used for farmed cervids, or for free-ranging cervids in limited geographical areas. Reduction in population density can be used where CWD is already present or as a preventative measure. Hunting provides one of the most effective strategies for reducing population densities for CWD management and surveillance activities. Targeted removal can reduce a specific subset

of an affected population (such as yearling males that are naturally dispersing from a CWD area). Testing and removal of CWD affected animals from a population would likely be appropriate only in limited situations. Vaccines and treatments for infected animals are not currently available. Restrictions on feeding or baiting of free-ranging cervids and regulations for the farmed cervid industry were examples of regulations of human behavior that may be used to control CWD. Habitat modifications that limit animal use of areas might be useful in dealing with environmental contamination and reducing the spread of disease to new areas. Federal and State agricultural and wildlife agency regulations that limit captive animal and carcass movements may help prevent the spread of CWD.

Other State and Provincial Management Actions.

CWD is considered endemic in an area of approximately 16,000 square miles in northeastern Colorado and southeastern Wyoming (Miller *et al.* 2000). Both states have recently adopted policies and plans for management of the disease. In addition, the disease has been found in free-ranging cervids outside the endemic area in Colorado, and in South Dakota, Nebraska, New Mexico, Saskatchewan, Illinois, and Wisconsin (Figure 1). Below are summaries of CWD management plans and policies that these states and provinces have adopted.

Multi-State Plan. A number of states, including Colorado, Wyoming, Nebraska, and South Dakota, that either have CWD or border states with CWD have been working to develop a comprehensive multi-state CWD management plan. The plan would serve as a guideline for development of state-specific plans but would not be binding on any signatory agency. The goals of the draft multi-state plan are to: 1) minimize the potential for CWD to spread; 2) manage infection rates within existing endemic areas according to each state's objectives; 3) eliminate the disease to the extent practicable when outbreaks occur in new areas; 4) support and conduct applied research that will expand knowledge of CWD; and 5) provide timely, complete, and accurate information about CWD to agency personnel and the public.

The plan recognizes that a more thorough understanding of CWD is needed for effective management of the disease and encourages states to participate in ongoing and future research.

The draft plan encourages states to develop detailed management plans for CWD-affected populations of deer and elk and recommends that artificial feeding and baiting should be banned or discouraged in affected areas.

The plan recognizes the importance of public hunting for managing deer and elk populations to reduce disease prevalence but stresses that disease management should take precedence over recreational hunting opportunities if they are in conflict. The plan further recognizes that it may be necessary for agency personnel or agents to cull animals for disease management and/or research and encourages states to establish the authority needed for such actions.

Because of potential health risks associated with consumption of deer and elk meat, the plan encourages state wildlife agencies to continue to work cooperatively with their public health agencies to monitor the health risks associated with CWD and to develop strategies for sharing current information about CWD with hunters in affected areas.

The plan recommends that states conduct surveillance to monitor the occurrence, distribution, and prevalence of CWD and the response to management. However, the plan recognizes differences between surveillance testing and food safety testing and recommends that state wildlife agencies not assume responsibility for food safety assurance. Rather states are encouraged to work cooperatively with public and private testing facilities to provide public hunters with information about available testing opportunities and costs to minimize the risk of consuming deer and elk with CWD.

The plan recommends that live free-ranging deer and elk not be translocated from CWD-affected areas except for research purposes and encourages states to exercise caution when considering translocation of deer and elk from areas not known to be affected.

The plan encourages state wildlife agencies to work cooperatively with their respective agriculture agencies to regulate the movement of captive deer and elk. The plan recommends that captive herds be monitored for at least 60 months before movements are authorized. Monitoring includes individual animal identification, annual reporting of herd additions and subtractions, and testing and reporting of all mortalities for CWD. The plan encourages states to develop uniform and consistent recommendations and regulations restricting the movement and disposal of carcasses of animals harvested in CWD-affected areas.

The plan also encourages states to provide ongoing training to all staff involved with CWD and to communicate with other state and federal agencies frequently to ensure coordination of actions. Lastly, the plan recommends that states continue to maintain effective communication with the general public, constituent groups, and the media about all facets of CWD.

Colorado. The policy approved by the Colorado Wildlife Commission (CWC) in 2002 recognizes CWD as a threat to the health of deer and elk populations throughout Colorado. Disease management objectives are to: 1) minimize the potential for disease spread beyond the current endemic area; 2) reduce levels of prevalence in the endemic area; 3) reduce the impact of disease on wild cervid populations; and 4) eliminate the disease from new areas outside of the endemic area (Colorado Division of Wildlife 2002). Colorado is preparing management plans for specific herd management areas. It is the policy of the CWC that, where conflicts occur, disease management will take priority over recreational hunting opportunity in these plans. The stated disease management objectives in these plans are to limit the distribution of CWD and to reduce prevalence to less than one percent in each Data Analysis Unit, and to less than two percent in each Game Management Unit (GMU). The rationale for these prevalence levels is based on predictive models. These plans call for expanded use of aggressive harvest and selective removal to achieve specific management objectives for disease containment and prevalence reductions. The feasibility of these objectives is unknown, but preliminary research findings suggest that targeted removal around CWD "hotspots" may be an effective management strategy (Ver Steeg 2002). The degree of population reduction required to reduce transmission of CWD is unknown. Current plans are to stabilize mule deer populations in endemic areas except in GMU 9, where the goal is to reduce the population to 50% of previous density and maintain it at that level to assess the effect of deer population reduction on prevalence rates. Public hunting will be the primary management tool for population control, but Wildlife Division staff is authorized to kill deer and elk for disease management and research, including the use of helicopters and fixed-wing aircraft.

Colorado's CWD management policy gives a priority to research to expand the knowledge of CWD. The policy recognizes that it is critical to provide hunters with the best scientific information available about CWD. The policy directs the Wildlife Division to work cooperatively with the Department of Agriculture to develop regulations to restrict the movements of captive cervids between commercial facilities. In addition, the Wildlife Division was directed to promulgate regulations to restrict the movement and disposal of carcasses and portions of carcasses harvested in GMUs where CWD is known to exist. Furthermore, the Wildlife Division was directed to continue to conduct CWD surveillance to monitor the occurrence and distribution of CWD based on an assessment of risk and available resources.

Artificial feeding of cervids has been banned in the endemic area of Colorado to lessen the probability of transmission (Williams *et al.* 2002a). Based on the high levels of infection observed in captive facilities, Williams and Young (1980) suggest that concentrations of deer and elk at artificial feeding and watering stations may facilitate transmission.

Colorado Division of Wildlife formed an international panel of experts to review their CWD management activities. This external panel generally agreed with Colorado's approach to management of CWD, but further recommended written plans for GMUs in the endemic area including methods to assess the effectiveness of CWD management objectives, more precise locational data from surveillance efforts, and continued research to understand CWD in free-ranging cervid populations.

Wyoming. Wyoming's management plan for CWD has three goals: 1) disease management; 2) applied research; and 3) public information (Wyoming Game and Fish Department 2002). Based on the current understanding of CWD in Wyoming's free-ranging deer and elk, the Game and Fish Department feels that eradication is not currently a realistic disease management objective within Wyoming's endemic area.

Wyoming is working to prevent the spread of CWD and, if possible, reduce the prevalence of the disease in its endemic area. The Game and Fish Department is conducting surveillance to determine spatial distribution and prevalence of CWD, and is conducting CWD research in cooperation with other state and federal agencies. The DNR plans to use all available communication tools to provide timely, complete, and accurate information about CWD to the public. The Game and Fish Department plans to continue to work cooperatively with the Wyoming Department of Public Health to evaluate human health risks of CWD.

Wyoming has chosen an adaptive management approach to allow flexibility in conducting disease management activities depending on the results of future research. The Game and Fish Department is preparing site-specific plans to assess the feasibility of using localized population reductions to reduce the risk of spread of CWD beyond the current endemic area. Surveillance will be conducted outside of the endemic area to promptly identify new disease areas. If a new focus area is detected, sufficient deer or elk will be killed by hunters or agency personnel to determine the extent of the infection and to attempt to eradicate CWD in the new focus area. Pending the results of research in Colorado and elsewhere, Wyoming will consider management actions to reduce or stabilize the prevalence in the endemic area. Other management strategies proposed to address their disease management goal include: 1) educating hunters about appropriate carcass movement and disposal to reduce the risk of spread; 2) restricting the translocation of live free-ranging or captive deer or elk; 3) discouraging private feeding of deer and elk within the CWD zone; and 4) maintaining the ban on captive deer and elk facilities.

Nebraska. Nebraska began surveillance for CWD in 1997. Since then, over 2,900 wild deer and 150 wild elk have been tested. Five mule deer and 10 white-tailed deer from four counties have tested positive (Morrison 2002). Game and Parks Commission policy for areas with CWD is to give priority to disease control over recreation. The management goal is to prevent the spread of CWD and eventually to eradicate it. Hunting regulations have been liberalized for areas where CWD has been found which allows hunters to assist with the control of the disease. Population management objectives are to reduce deer herds in these areas by at least 50% (Bruce Morrison, Nebraska Game and Parks Commission, pers. comm.). Removal operations will continue to be conducted in areas where CWD positive animals are found. The Commission has banned hunting over bait and the translocation of wild cervids within Nebraska. Commission policy is for CWD research to receive priority for funding and for full and complete public disclosure of all information and data.

South Dakota. CWD has been confirmed in two free-ranging white-tailed deer and one free-ranging elk in South Dakota (Fowler 2002a, VerSteeg 2002). Surveillance efforts have intensified, and population management goals for units at risk for CWD were changed from encouraging maximum deer populations within landowner tolerance to reducing deer density (Fowler 2002b). Harvest permits have increased significantly in these units (Chuck Schluteter, South Dakota Game, Fish and Parks, pers. comm.).

Saskatchewan. CWD has been confirmed in seven wild mule deer in Saskatchewan since 2000. Disease eradication is the stated management goal and population reduction efforts have begun in a 400 square mile area with the goal of reducing deer populations by 60% (Adam P. Schmidt, Saskatchewan Environment and Resource Management, pers. comm.). Quotas for mule deer permits in these areas have increased significantly. In addition, removal activities by agency staff have been conducted within four miles of the known positive deer.

New Mexico. New Mexico has been conducting surveillance for CWD for a number of years. A mule deer collected from the White Sands Missile Range in southcentral New Mexico tested positive for CWD in 2002 (New Mexico Department of Game and Fish 2002). In response, New Mexico closed its borders to the import of cervids and is continuing to sample hunter-harvested deer and elk to determine the distribution and prevalence rate of CWD in the state

Illinois. Illinois has confirmed seven cases of CWD disease in Illinois. Illinois expanded surveillance efforts regarding CWD in 2002 and created a joint task force with the Departments of Natural Resources and Agriculture following the CWD outbreak in southern Wisconsin. About 4,000 samples of hunter-harvested deer were taken in 36 counties around Illinois during the 2002 firearm deer season Nov. 22-24 and Dec. 5-8. Tests have been completed on about 1,450 samples, with seven positive results to date.

In response to CWD, Illinois has limited the importation of hunter-harvested deer or elk, restricted the importation of live animals, and banned feeding of wild deer. Illinois is waiting for completion of testing their remaining samples before further formulating their management response to CWD.

Thirty-one states are currently in the process of developing new and/or additional CWD regulations in response to these CWD management goals. Forty-six states conduct CWD testing of wild deer and elk, and two additional states are in the process of developing surveillance programs. In addition, at least 26 states and two Canadian provinces do not allow baiting of deer and elk. At least three states have enacted restrictions or are in the process of developing rules to restrict or ban baiting. At least 12 states do not allow the feeding of deer or elk, or restrict the use of feed. Nine states and one Canadian province have put restrictions on the importation of hunter-harvested deer and elk parts and six states are discussing similar bans.

Federal Environmental Assessments.

The United States Department of Agriculture—Animal and Plant Health Inspection Service (USDA – APHIS) developed an Environmental Assessment (EA) in the Summer of 2002. The EA outlined the role of the Federal Government in CWD management. The EA reported that the primary Federal role in the CWD program would be to provide coordination and assistance with research, surveillance, disease management, diagnostic testing, technology, communications, information dissemination, education, and funding for the State CWD Program. In addition to this more general plan for CWD management, the Wildlife Services branch of USDA-APHIS is drafting an EA outlining plans to reduce cervid damage in Wisconsin and the role that agency may play in assisting the DNR with CWD management in Wisconsin.

History of CWD in Wisconsin.

The Wisconsin Department of Natural Resources (DNR) began active CWD surveillance of hunter-harvested deer in 1999. Through 2001 approximately 1,100 deer were sampled throughout the state. In February 2002, the DNR was notified that three male deer harvested from Deer Management Unit 70A in western Dane County tested positive for CWD. In response, an interagency team was formed consisting of the DNR, Agriculture, Trade, and Consumer Protection (DATCP), and Health and Family Services (DHFS). A 12-mile radius surveillance area was designated that was centered on the three index cases. During March and April, 516 deer were collected from within the surveillance area of which 15 (2.9%) tested positive for CWD using immunohistochemistry (IHC) assays of brain stem and lymph node tissues. However, prevalence was not uniformly distributed in the surveillance area.

The Wisconsin DATCP began a voluntary CWD monitoring program for farm-raised deer in 1998. Until 2002 there were approximately 40 captive herds enrolled in the program, the majority of which were elk herds. The emergency rule adopted by DATCP in April 2002 greatly increased CWD testing and monitoring. The emergency rule required monitoring all captive herds for CWD by farmers that moved live deer or elk off a farm. The emergency rule also required testing of all deer and elk carcasses removed from farms. By December 2002, there had been over 1700 CWD tests done on farmed deer in Wisconsin and there were 260 herds fully enrolled in the state's CWD monitoring program.

A male white-tailed deer from a deer farm in Portage County tested positive for CWD in September of 2002 when it was sampled in compliance with the rule requiring testing of all carcasses if any part of the carcass is to leave the farm. This finding triggered an investigation which resulted in identifying a CWD-positive female white-tailed deer on a Walworth County farm that same month. Another deer, assumed to have escaped from the same Walworth County farm in April 2002, was shot near the farm and tested positive for CWD in October 2002. All of the deer in the Walworth County herd were killed and tested for CWD in December 2002 and four additional CWD infected deer were identified. The CWD positive deer on both of these farms appear to have originated on a second Walworth County farm. All three farms were quarantined in September 2002 and the two remaining farms will stay under quarantine until they are depopulated.

As of January 31, 2003 36% of the statewide tests results had been returned that were collected during the 2002 and early 2003 statewide CWD surveillance testing. A total of 38,764 total samples were taken (11,434 in the eradication zone, 5,808 in the Management Zone, and 21,470 in the remainder of the state;

4½ miles out from any additional positive deer. The deer population goal for the EZ was set to zero. The IHZ was slightly larger than the EZ with borders defined on state and county roads. The deer hunting season for the IHZ in 2002 was extended to a 4½ -month archery season and a gun season that ran from late October through January. The CWD Management Zone extended out from the center of the EZ approximately 40 miles. Population goals in the Management Zone were established at 10 deer per square mile of deer habitat. The gun season in the Management Zone consisted of three segments: one in late October, one from late November through mid-December, and one during the Christmas-New Years period. The 2002 archery season ran from mid-September to early January. An unlimited number of earn-a-buck (must harvest an antlerless deer prior to harvesting an antlered deer) permits were offered for both the IHZ and the Management Zone in 2002.

The emergency rule specified the conditions under which DNR staff can shoot deer from vehicles and aircraft. The rule identified deer within the EZ to be causing a nuisance and authorizes the DNR to issue permits to landowners and their permittees to shoot deer during periods defined by the DNR throughout the year. In addition, the emergency rule prohibited the use of bait for deer hunting and the artificial feeding of deer to reduce the probability of CWD transmission (see section on Baiting and Feeding). The rule also expanded firearm options and deer seasons in state parks, and authorized the issuance of replacement permits if hunters shoot diseased deer. The rule further specified transportation, registration, and disease sampling requirements for harvested deer.

These regulations were developed following discussion with the public and many organizations at meetings across the state and following review of public input on alternatives presented in a questionnaire at meetings and on the internet (Appendix D). Over 3,000 questionnaires were completed and returned. Focus groups were held with landowners and hunters from the affected area to get additional public input. DNR recommendations followed this input with a few modifications based on experience with deer herd reduction regulations. Conflicts with other land uses were considered; however, control of CWD through herd reduction was given a higher priority than conflicts with other recreational uses.

During the 2002 CWD Management Zone hunts approximately 41,000 deer were harvested. As of January 24, 2003 approximately 11,000 deer have been killed, but the seasons in the IHZ were still in progress.

Wisconsin Act 56, as of January 1, 2003, moved the authority to regulate deer farms from the DNR to the DATCP, however deer farm fencing inspection authority still rested with the DNR. Therefore, new fencing standards were created with an emergency rule until a permanent rule could replace it. While incorporating many of the deer farm fence standards from ch. NR 16, Wis. Adm. Code, the rule increased the required height of new fences from 8-10 feet. It also phased in a requirement that deer farms be double fenced unless the deer farm is enrolled in the CWD herd monitoring or herd surveillance program.

Summer 2002 Deer Removal.

Landowners within the EZ were issued permits for shooting deer during four one-week periods in June-September 2002. Landowners and agency shooters collected over 1,500 deer during these summer shooting periods. Thirty-one deer collected during June – September tested positive for CWD.

Actions Taken by Other Wisconsin State Agencies to Control CWD.

DATCP Captive Cervid Regulations

Currently in Wisconsin there are 575 white-tailed deer farms, 272 elk farms, and 100 farms that have deer species such as red deer, fallow deer, sika deer, and reindeer registered with the Wisconsin DATCP (Figure 4). These farms represent about 35,000 cervids. They are widely scattered throughout the state. Shortly after the discovery of CWD in free-ranging white-tailed deer in Wisconsin in spring 2002, DATCP wrote emergency rules to restrict importation of cervids. These rules required that only cervids from herds that had enrolled in a state-sponsored CWD monitoring program or the equivalent for five years may enter the state. Herds must have never had any signs of CWD, must have had no connection to any CWD infected herds. In addition, owners must have kept accurate records of all transactions, deaths, and causes of death in the herd for the last five years.

The emergency rules require all cervid farms that move live animals off the farm to be enrolled in the CWD monitoring program. Monitoring for CWD in captive herds relies on testing animals that die, are harvested, or otherwise removed from the herds over several years. Provisions of the CWD monitoring program are:

- All cervids one-year old and older must have individual official identification.
- The owner must submit an annual herd census, records of animals moved off the farm or added to the farm, records of all deaths, and the CWD test results of those deaths, to account for all of the animals in the herd from year to year.
- All cervids that die or are killed for any reason and are at least 16 months old must be tested for CWD.
- A veterinarian must provide an annual letter about the CWD status of the herd.

Those herds that do not move any live animals off the farm are required to test any animals that are 16 months or older that die for any reason if the carcass or parts of the carcass leave the farm. This includes CWD testing of animals harvested by hunting if any part of the animal will leave the farm (including antlers or hides). It also includes any animals harvested where any meat will leave the farm. If the carcass must be removed from the property for disposal (e.g., burial) the animal must be tested for CWD.

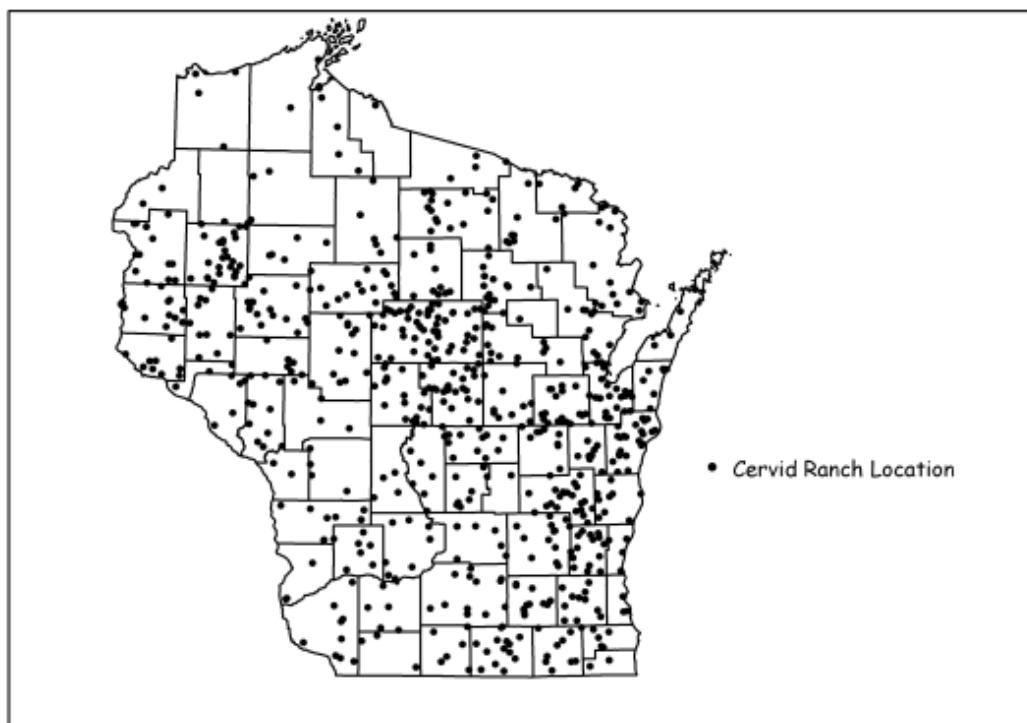


Figure 4. Location of all Licensed Captive Cervid Facilities in 2000.

In fall 2002, CWD was detected on two game farms, one in Portage County and one in Walworth County. These farms plus several others who traded deer with the affected farms were placed under quarantine. At this time, work is progressing to depopulate three captive farms.

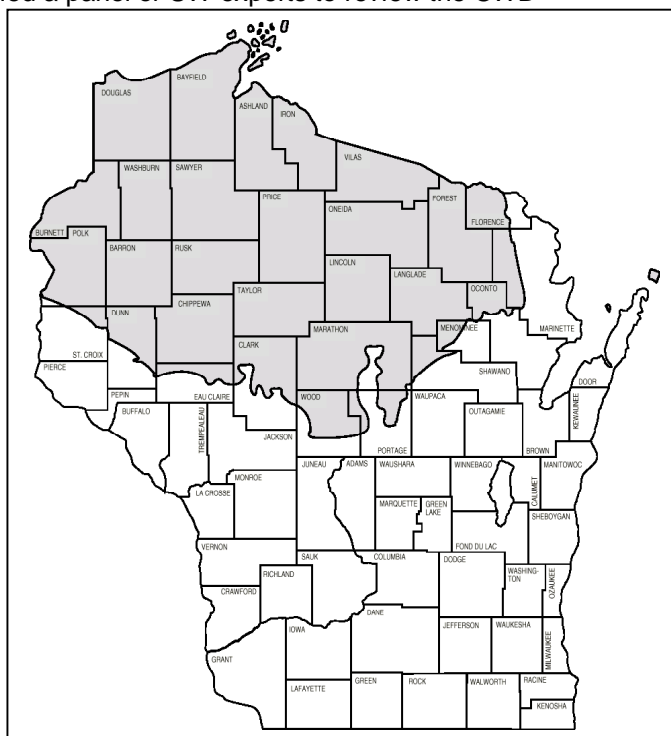
In December 2002, DATCP Board approved a set of permanent rules that were similar to the emergency rules adopted in the spring. The permanent rules were strengthened by requiring owners of farm-raised deer to report all escapes within 48 hours and to notify a certified veterinarian within 24 hours of observing any signs or symptoms of CWD. The permanent rules further required that every farm-raised deer over 16 months of age that dies on the herd premises be tested for CWD.

Currently, there is no scientific evidence that CWD poses a risk to human health. However, there are aspects of prion transmission that remain unknown, and no one can guarantee that absolutely no risk exists regarding human consumption of animals that have CWD. The Department of Health and Family Services (DHFS) has been working closely with the DNR to help provide hunters and venison consumers with information about potential human risks and ways to minimize those risks that is accurate, current and understandable so that prospective hunters may make informed decisions.

University Involvement.

The University of Wisconsin (UW) provided resources to assist with peer review of CWD management plans, computer modeling, CWD genetics, and deer movement and behavior research, and public outreach and education activities. They assembled a panel of UW experts to review the CWD management plans that were being proposed. An individual-based, spatially explicit model was developed at the UW that predicted the likely results from using different management approaches to control and eradicate CWD. This model was used as one of the tools to assist in the selection of a strategy for controlling the disease. Currently, UW research projects investigating genetic disease resistance and genetic strain typing of CWD prions and dispersal and social behavior of deer are ongoing. UW staff facilitated town meetings with the public and provided numerous educational activities regarding CWD.

In 1983, the 7th Circuit Court of Appeals determined that the Wisconsin Bands of Ojibwa Indians (referred to hereafter as Chippewa Tribes) retained their rights to hunt, fish, and gather living natural resources, including the right to hunt deer, by the Treaties of 1837 and 1842. These rights apply to public lands within the ceded territories of northern Wisconsin, and include all or parts of 63 deer management units



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(DMUs) within all deer management regions except the southern portion of the state (Fig. 5). In 1990, Judge Barbara Crabb ruled that the system the tribes use to monitor and limit harvest was adequate to protect the resource and the tribes were determined to be self-regulatory.

Among the rulings of the Court was that "the tribal allocation of treaty resources is a maximum of 50% of the resource available for harvest" (Great Lakes Indian Fish and Wildlife Commission 1991). For a discussion of tribal harvest allocation and deer seasons see Appendix F. The Great Lakes Indian Fish and Wildlife Commission (GLIFWC) is an inter-tribal, natural resource management organization that acts under delegated authority from its member tribes in the implementation and protection of treaty rights in the ceded territories. GLIFWC biologists participate with the Wisconsin DNR in setting the deer harvest quota each spring. In addition, GLIFWC assisted with the statewide CWD surveillance effort in fall 2002.

None of the ceded territory falls in the southern portion of the state where the current eradication and herd reduction zones are located. The DNR would seek joint tribal participation if CWD is discovered in the ceded territories and would require the implementation of CWD control measures.

Wisconsin CWD Management Plan

A CWD Management Plan was developed in response to the discovery of CWD in Wisconsin. The goal for CWD management in Wisconsin is to minimize the negative impact of CWD on wild and captive cervid populations, the state's economy, hunters, landowners, and other people dependent upon healthy wild and farmed populations of deer and elk. This goal will be accomplished by five major actions: 1) surveillance, 2) human health protection, 3) CWD research, 4) communications, and 5) disease control.

CWD Surveillance.

Disease surveillance is key to the implementation of the management plan. Intensive surveillance sampling was conducted during fall 2002, but continuing surveillance will be a critical part of the CWD management program in the future. Surveillance strategies were developed for three distinct regions in Wisconsin: the current EZ/IHZ, the current CWD management zone, and the rest of the state. Surveillance sampling relied on hunter-killed deer for most samples. In some areas of the state, hunter-killed deer were supplemented by deer killed under deer damage shooting permits or collections of car-killed deer. In addition, DNR staff responded to any report of sick deer in the state and any CWD suspect deer are tested for the disease.

Disease surveillance was conducted in the CWD affected area (EZ and IHZ) to determine the prevalence of disease. Tissue samples were collected from every deer killed in the EZ. Sample collection during the fall hunting seasons began on September 14, 2002 with the beginning of the bow deer season and continued until the end of the bow and gun seasons on January 31. The number of samples collected depended upon hunter success. In addition, efforts were made to collect and test any deer exhibiting clinical signs of CWD (targeted surveillance). Estimates of CWD prevalence over time and geographic extent of the infection are needed to evaluate the effectiveness of disease control activities. This information is also critical for targeting control activities to areas with the highest level of prevalence. Disease surveillance also supports research investigations to help identify transmission mechanisms and to model disease spread on the landscape.

Sampling for CWD in the CWD Management Zone was designed to achieve a comprehensive spatial sample of deer and to reach a high probability (e.g., 99%) that CWD will be detected during early stages of a disease outbreak. To reach these goals several years of surveillance efforts and evaluation will likely be required. In the CWD Management Zone 500 or more deer were sampled from each Deer Management Unit in 2002. In these units the DNR limited sample collections to deer that were at least 1½-years old. Sample collection took place during the October 24-27 early gun deer season and again on the opening weekend of the regular gun deer season (November 23-24). All samples were submitted for testing as soon as they were processed.

In the rest of the state, the primary objective for surveillance was early detection of CWD new areas. Surveillance was designed to achieve a systematic random spatial sample of deer and to reach a high probability (e.g., 99%) that CWD will be detected during early stages of a disease outbreak. A sample of 500 deer was the goal for each county. In some areas of the state, where the deer population is small or the deer kill is insufficient to get a statistically reliable sample, several counties were grouped into a single sampling unit. Sample collection occurred during October 24-27 in those counties with a Zone-T season. Sampling also occurred on the opening weekend of the gun deer season (November 23-24). Both targeted surveillance of suspect deer and collections of deer heads from hunter harvested deer were used. In these units the DNR limited sample collections to deer that were at least 1½-years old. All samples were submitted for testing as soon as they could be processed. Early detection is important for timely response to new disease affected areas.

Sample collection sites for all three areas of the state have been selected based upon the volume of deer processed at these sites in previous deer seasons (Figure 6.). At all collection sites, the head of the deer were removed and tagged with an identification number and a data collection form was completed for each deer sampled. Deer heads were transported to one of five regional processing centers where tissue samples were extracted and data entry of all collection data occurred. The regional processing centers were located in Park Falls, Green Bay, Eagle, Black River Falls, and Black Earth. The processing centers, with the exception of Black Earth, were operational only during the days that sample collection occurred. Black Earth served as the statewide tissue-staging site before shipping the samples for testing. The Black Earth site is operational for a longer period of time to handle the continuous submission of deer heads from the EZ during September 14-January 31.

The Wisconsin Veterinary Diagnostics Lab (WVDL) processed most of Wisconsin's surveillance samples. Any samples in excess of WVDL's capacity were sent to USDA's National Veterinary Services Lab in Ames, IA for processing through their national system of CWD testing labs.

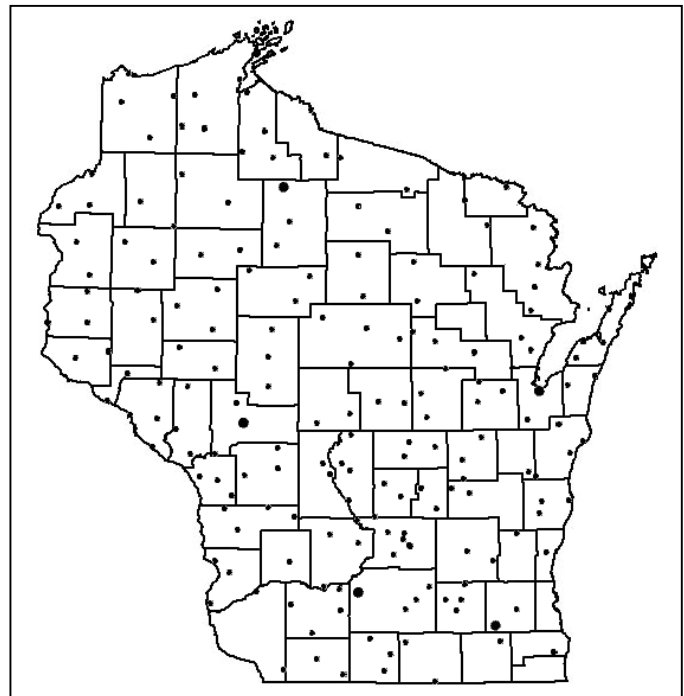


Figure 6. Location of CWD Sample Collection Sites (•) and Regional Processing Centers (●) during October 2002 surveillance testing.

Human Health Protection.

Human health protection will primarily be conducted by the Department of Health and Family Services by monitoring cases of CJD in an effort to determine if there is any relationship with CWD and providing information to hunters and venison eaters on the safety of eating venison.

CWD Research.

The DNR, in cooperation with the University of Wisconsin and the USGS-National Wildlife Health Center, is implementing a large research program to expand the scientific information needed for managing CWD in Wisconsin and to evaluate the effectiveness of the CWD management program. This research program incorporates studies on disease dynamics, deer ecology, and human dimensions. The disease dynamics study will determine the geographic distribution and prevalence of the disease, estimate transmission routes and methods of spread, and evaluate age/sex and genetic relationships and genetic resistance to CWD. The deer ecology study will focus on population dynamics, movements and behavior of deer and will evaluate the effectiveness of the CWD management program. The human dimensions study will investigate perceptions of human risk, explore attitudes of hunters, landowners, and the public about CWD management, and estimate the economic effects of CWD.

Communications.

Since the discovery of CWD in Wisconsin, the DNR has attempted to provide timely, complete, and accurate communication with the citizens of the state a priority component of its management plan. The DNR has used all available communication tools in this effort including news releases, television appearances, radio interviews, brochures, handouts, public meetings, and the Internet. A web site has been established that is updated weekly with new information and test results from samples submitted by hunters.

Disease Control.

At the present time, Wisconsin's CWD Management Plan assumes that the disease is limited to southwest Wisconsin. As outlined in the National CWD plan, the best management strategies for this situation is to: 1) depopulate the deer herd in the known affected area; 2) reduce deer populations around the affected area to establish a barrier to prevent the spread of CWD outside the affected area; and 3) ban baiting and feeding to limit the transmission of the disease.

In the rest of Wisconsin, the spread of CWD is minimized by: 1) Zone-T seasons where deer populations are above established goals; 2) prohibition of baiting and feeding of deer; and 3) educational efforts on the proper disposal of deer carcasses.

The following three sections assess the effects of these disease control actions proposed in the rule package: depopulation of deer in an EZ, herd reduction in a HRZ, and a statewide ban on baiting and feeding deer. Each section assesses the need for this action and then evaluates the tools proposed to accomplish the action.